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Smoke Inhalation

These discussions are selected from the weekly staff conferences in the Department of Medicine, University of California, San Francisco. Taken from transcriptions, they are prepared by Drs. David W. Martin, Jr, Professor of Medicine, and James L. Naughton, Assistant Professor of Medicine, under the direction of Dr. Lloyd H. Smith, Jr, Professor of Medicine and Chairman of the Department of Medicine. Requests for reprints should be sent to the Department of Medicine, University of California, San Francisco, School of Medicine, San Francisco, CA 94143.

DR. SMITH:* *At this time of year it is customary to invite our chief residents to present Medical Grand Rounds. This enables us to express publicly our appreciation for the superb leadership they have provided in our teaching and patient-care program. Dr. Diana Coleman has chosen the topic of smoke inhalation for discussion.*

Case Presentation

Dr. COLEMAN:† A 33-year-old, previously healthy man was admitted to hospital because of an inhalation injury sustained during a fire in his apartment. The patient had awakened to find his bedroom filled with smoke and his living room ablaze. He attempted to escape but was found semiconscious near the closed patio door in his bedroom. The patient was removed from the fire. Oxygen was administered by face mask, and he was brought to the emergency room approximately 20 minutes after being found.

In the emergency room, he was conscious, coughing up sooty material and complaining hoarsely of shortness of breath. Blood pressure was 110/70 mm of mercury, heart rate 130 beats per minute and respiratory rate 18 breaths per minute. There was no pulsus paradoxus. The skin was sooty but there were no cutaneous burns; there was no cyanosis or cherry red coloration. Examination of the head, eyes, ears, nose and throat showed pronounced conjunctival injection, soot in the mouth and nose, and erythematous oral and nasal mucous membranes. There was no stridor. Auscultation of the lungs disclosed occasional expiratory wheezes. The remainder of the physical examination showed no abnormalities.

Results of laboratory tests were as follows: hemoglobin 15.6 grams per dl, hematocrit 46.2 percent and leukocyte count 12,600 per cu mm. The serum sodium level was 138, potassium 4.6, chloride 100 and bicarbonate 22 mEq per liter; blood urea nitrogen was 14 and creatinine 1.1 mg per dl. Results of the SMA-12 analysis were within normal limits. A 12-lead electrocardiogram showed sinus tachycardia at a rate of 120 beats per minute. There were nonspecific ST segment and T wave abnormalities in all leads. A roentgenogram of the chest showed no abnormalities. Arterial blood gas measurements obtained while the patient was breathing oxygen through a nonrebreathing mask were pH 7.33, partial pressure of carbon dioxide 30 mm of mercury, partial pressure of oxygen (Po_2) 378 mm of mercury, calculated oxyhemoglobin saturation 99 percent and measured carboxyhemoglobin (COHb) saturation 45 percent. Sputum Gram stain showed 2+ polymorphonuclear leukocytes and carbon debris.

During the next ten hours the patient's dyspnea,

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ABBREVIATIONS USED IN TEXT

P_{O_2} = partial pressure of oxygen
COHb = carboxyhemoglobin

hoarseness and wheezing worsened. Transnasal fiberoptic bronchoscopy showed substantial hypopharyngeal and laryngeal edema, and a nasotracheal tube was placed. Aminophylline was given intravenously and terbutaline subcutaneously, and the patient's wheezing lessened moderately. Oxygen therapy was continued and four hours after admission the COHb saturation was below 10 percent.

By the third hospital day, the laryngeal edema was decreased; however, suctioning yielded not only tenacious sputum but apparent bronchial casts as well. A temperature developed to 39.5°C (103.1°F). Arterial blood gas measurements showed increasing hypoxia, and a roentgenogram of the chest showed partial collapse of the right middle and right lower lobes, with pulmonary infiltrates. At bronchoscopy, plugs of mucus and debris nearly occluding the right middle and right lower segmental bronchi were seen. Sputum Gram stain showed 4+ polymorphonuclear leukocytes and both Gram-positive and Gram-negative organisms. The patient was treated with intravenously given penicillin and tobramycin. Sputum culture showed mixed oronasal flora and tobramycin administration was discontinued. The right middle and right lower lobes reexpanded fully and the patient's temperature returned to normal. By the eighth hospital day the patient's tracheal secretions were diminished considerably, and he was extubated.

At the time of discharge on day 14 the patient had no respiratory complaints. Findings of the physical examination and roentgenogram of the chest were normal as were arterial blood gas measurements.

Introduction

Fires causing large numbers of deaths have been recorded throughout this century in the United States. In 1903 a total of 602 people died in the Iroquois Theatre fire in Chicago. In 1930 a fire at an Ohio penitentiary killed 320, and 165 patrons died in Southgate, Kentucky, in 1977, when the Beverly Hills Supper Club burned. In November 1980 the fire at the MGM Grand Hotel in Las Vegas resulted in 84 deaths. Spectacular fires such as these understandably receive a great deal of attention by the press: a large number of people are at risk and major structures are threatened. However, the National Fire Protection Association (NFPA) estimates that hotel and motel fires accounted for only 0.4 percent of the 2,845,000 fires nationwide in 1979, whereas fires in other residential dwellings accounted for 25 percent of the total.[1] Approximately 140 civilians died and 1,225 were injured in the 11,500 hotel and motel fires in 1979. In contrast, 72 percent of the 7,780 total civilian deaths and 61 percent of the 31,325 total civilian injuries in the United States in 1979 resulted from the 710,000 residential fires that occurred that year. It is estimated that 55 percent to 80 percent of deaths due to fires result from smoke inhalation.[2]

Most of the morbidity and mortality resulting from the inhalation of smoke is a result of either systemic toxicity or complications from local respiratory tract injury. This paper will review the history and pathogenesis of smoke inhalation injury, the systemic illness resulting from inhalation of toxic gases, and the local effects from such inhalation on the respiratory system. Specific attention will be focused on carbon monoxide intoxication, the diagnosis of local injury to the respiratory system, the problem of upper airway obstruction, and the initial evaluation and management of patients with smoke inhalation injury.

Historical Background

The lethal qualities of smoke were appreciated as early as the first century AD, when Pliny the Elder recorded that prisoners were executed by placing them in cages suspended over green wood fires.[3] Medical attention was focused on the clinical significance of smoke inhalation and subsequent respiratory tract injury in 1942, when 489 persons died in the Cocoanut Grove Nightclub fire in Boston.[4,5] Of the 181 survivors who required hospital care, 170 were admitted to either the Massachusetts General Hospital (MGH) or Boston City Hospital.[4,5] Of the 39 people admitted to MGH, injuries to the respiratory tract developed in 36, and 7 of the victims died. In 97 of the 131 patients at Boston City Hospital, involvement of the respiratory tract developed, and 39 died. From these experiences it became apparent that smoke inhalation and subsequent injury to the respiratory tract were important causes of morbidity and death.

More recently, at the MGM Grand Hotel fire

in Las Vegas, more than 5,000 people were at risk of smoke inhalation or thermal injury. The NFPA estimated that at least 679 people were injured.[6] Within a few hours, more than 600 of the injured people received care at several hospitals; of these, more than 300 required hospital admission. Of the 84 deaths, 79 were a result of smoke inhalation. Most of the victims were found on the 20th through 25th floors of the hotel, far above the fire, which was confined to the first floor. They were often found clustered in elevator lobbies and in enclosed stairwells, two areas that filled with dense smoke during the fire.

Pathogenesis of Inhalation Injury

Smoke is a suspension of small particles in hot air and gases. The composition varies with the materials that undergo thermal degradation and combustion, the temperature of the process and the concentration of oxygen available.[7] The injury produced depends on the composition of the smoke and the duration of exposure.[8]

Smoke inhalation causes injury by thermal, chemical and hypoxemic mechanisms and results in systemic injury as well as damage to the upper and lower respiratory systems. Injury to the upper respiratory system is caused by both thermal and chemical processes. Injury to the lower respiratory system is usually due to chemical mechanisms. Systemic illness results from both chemical and hypoxemic processes.

Thermal Injury

In a series of experiments in 1944, the respiratory tracts of dogs were exposed to oven-heated air, flame and combustion products from a blast burner, and steam.[9] To prevent the animals from dying from obstructive asphyxia due to pharyngeal and laryngeal edema, the hot gases and combustion products were delivered through an insulated transoral cannula passed through the larynx directly to the trachea. The temperature of the inhaled mixtures was monitored by thermocouples located at the laryngeal end of the cannula and at the carina. The temperature of the heated air and blast flame mixtures ranged from 250° to 550°C at the laryngeal end of the cannula, and cooled to less than 100°C by the time the carina was reached. The temperature of the inhaled steam fell less dramatically from 100°C as it traversed the same path. Pathological examination of the tracheobronchial tree and pulmonary parenchyma showed tracheitis but no significant lung injury except when steam was used. This suggested that thermal injury alone played little role in respiratory tract injury below the larynx unless an agent with a high specific heat, such as steam, was involved.

TABLE 1.—*Common Toxic Products of Combustion**

Substance	*Toxic Products of Combustion*
Polyvinyl chloride	Hydrogen chloride, phosgene, chlorine
Wood, cotton, paper ..	Acrolein, acetaldehyde, formaldehyde, acetic acid, formic acid
Petroleum products ...	Acrolein, acetic acid, formic acid
Nitrocellulose film	Oxides of nitrogen, acetic acid, formic acid
Polyurethane	Isocyanate, hydrogen cyanide
Melamine resins	Ammonia, hydrogen cyanide

*Adapted from Fine et al.[10]

Chemical Injury

Subsequent experiments have confirmed the impression that thermal injury per se does not commonly cause injury to the lower respiratory tract, but that most of the damage is produced by toxic products of combustion. Thermal degradation and combustion of both man-made and natural materials in the environment result in the generation of numerous toxic chemicals.

Polyvinyl chloride is a synthetic compound commonly used in modern building furnishings and fixtures. It does not ignite until it reaches 475°C; however, from 225° to 475°C it loses 60 percent of its weight by thermal degradation. More than 75 known toxic products are formed in the process, including hydrogen chloride, phosgene and chlorine.[7] Combustion of other common materials results in the generation of numerous organic acids, aldehydes and other noxious gases, including oxides of sulfur and nitrogen (Table 1).[10]

Aldehydes, especially acrolein, cause irritation of the exposed mucous membranes, pulmonary edema and death.[8] Irritant gases such as ammonia and sulfur dioxide combine with water present in the mouth and tracheobronchial tree to form damaging alkalies and acids.[11] Soot aerosols absorb toxic gases such as hydrogen chloride and transport them to alveolar areas where they cause further damage.[12]

Mucosal edema, bronchorrhea, and sloughed mucosa caused by chemical injury increase airway resistance and cause obstruction to airways. Inhaled soot particles, in addition, cause broncho-

constriction, possibly by stimulation of irritant receptors in the airways.[13]

Chemical injury resulting from smoke inhalation depresses pulmonary defense mechanisms, which are further compromised by any associated cutaneous burns.[14-19] Bacterial pneumonia is, therefore, a common complication of inhalation injury.[20-25]

Hypoxemic Injury

Severe hypoxemia results most often from either asphyxia or carbon monoxide poisoning. Fire in a closed area progressively consumes the available oxygen, and various products of combustion dilute the concentration of the remaining oxygen, diminishing its partial pressure. This subsequently leads to hypoxia. Carbon monoxide intoxication is one of the most frequent immediate causes of death from smoke inhalation.[26]

Systemic and Local Respiratory System Effects

Illness resulting from smoke inhalation can be divided into two major processes: the systemic effects of toxic gases such as carbon monoxide that are absorbed from the respiratory tract and the direct local effects of the inhaled substances on the respiratory system.

Systemic Injury

Carbon Monoxide

Carbon monoxide, a ubiquitous product of incomplete combustion, causes death as a result of its systemic effects. In autopsy studies conducted in fire victims in Maryland and New York City, carbon monoxide caused or contributed to 80 percent of the fatalities that occurred within 12 hours of injury.[26,27] This colorless, odorless gas was cited as the sole factor responsible for failure to escape from a fire nearly 30 percent of the time, and, in combination with alcohol, it was cited an additional 34 percent of the time.[26]

Carbon monoxide produces its toxic effect by three mechanisms: It decreases oxygen transport by hemoglobin; it decreases oxygen unloading at tissues, and it impairs the use of oxygen by tissues. Because of the increased affinity of carbon monoxide for hemoglobin, the amount of hemoglobin available to bind oxygen is decreased and the oxygen-carrying capacity of the blood is reduced (Figure 1).[28] At a COHb saturation of 50 percent, the oxygen content of arterial blood at a partial pressure of oxygen (Po_2) of 100 mm of mercury is approximately 50 percent of normal. This is similar to that seen in an otherwise normal person whose hemoglobin concentration has been reduced by 50 percent. However, despite the similar oxygen contents, the patient with carbon monoxide

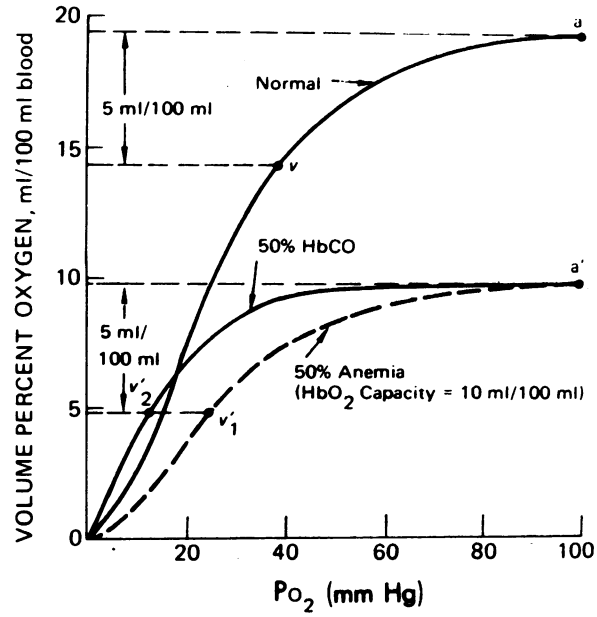

Figure 1.—Arterial blood oxygen content plotted against partial pressure of oxygen (Po_2) in a normal person, a person with 50 percent carboxyhemoglobin (COHb) and a person with anemia and a 50 percent reduction in hemoglobin concentration (HbCO). (Reproduced with permission from National Academy of Sciences.[28])

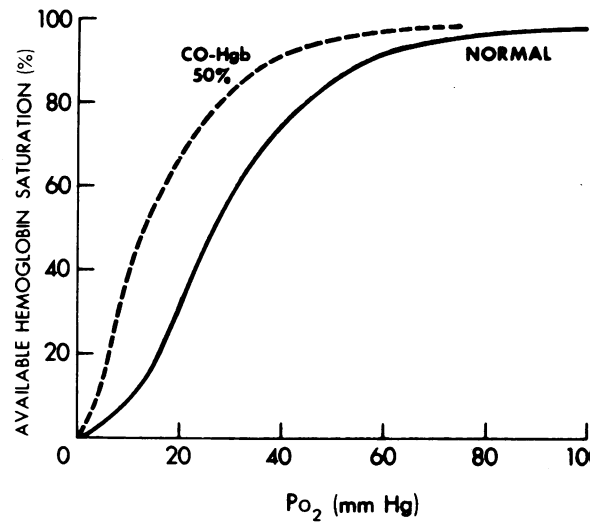

Figure 2.—Oxyhemoglobin equilibrium curve in a normal person and a person with 50 percent carboxyhemoglobin (CO-Hgb) (Po_2 = partial pressure of oxygen). (Reproduced with permission from Hinshaw & Murray.[29])

intoxication has less capacity for oxygen unloading at the tissues. This occurs because in the presence of COHb, the oxyhemoglobin dissociation curve is shifted to the left (Figure 2).[29] Therefore, for any given partial pressure of oxygen, more of the available hemoglobin remains saturated and less oxygen is available to the tissues. In addition, carbon monoxide may be directly toxic at the cellular level by its action on the cytochrome enzyme systems.[30]

The affinity of carbon monoxide for hemoglobin is approximately 200 times greater than oxygen's affinity for hemoglobin.[31,32] Therefore, even brief exposure to carbon monoxide can result in a significant COHb saturation. Endogenous production of carbon monoxide during metabolism of hemoglobin or other tetrapyrroles usually results in COHb saturation of less than 0.7 percent.[32] Mean COHb saturation in smokers of one pack of cigarettes a day has been reported at 1.9 percent and in smokers of two packs a day at 3 percent.[32] Smoke at fire scenes can contain carbon monoxide in concentrations that vary from 0.1 percent to 10 percent (1,000 to 100,000 ppm).[33] It has been calculated that a 30-second exposure to 1 percent carbon monoxide during heavy exertion such as might be involved in fighting or escaping from a fire (30 liters per minute alveolar ventilation) would result in a COHb saturation of 7.5 percent, and a two-minute exposure to the same concentration of carbon monoxide would result in a saturation of 30 percent.[33] A two-minute exposure to 2 percent carbon monoxide under the same conditions would produce a lethal COHb saturation of 60 percent (Table 2).

Signs and symptoms of carbon monoxide poisoning vary with the amount of COHb present (Table 3) and offer no pathognomonic clues for diagnosis.[34] Findings on examination of the skin may occasionally aid in the diagnosis. The classic cherry red skin color associated with the presence of COHb was observed in only 14 of 105 patients in one clinical series.[35] Cyanosis was a much more common finding, occurring in 43 of the 105 patients. Irregularly bordered erythematous skin patches, vesicles and bullae have also been noted.[36]

Carbon monoxide intoxication should always be suspected in persons found in fires. The diagnosis can only be inferred from routine arterial blood gas measurements. The measured oxygen content is always diminished; however, this determination is not routinely available. The Po_2 reading is usually normal, even in the patient with significant carbon monoxide intoxication. Therefore, the calculated oxyhemoglobin saturation is also normal. When present, an associated acidemia and elevated anion gap further raise the suspicion of carbon monoxide intoxication.[37] The definitive diagnosis relies on the measurement of blood COHb saturation. Because the COHb saturation begins to decrease as soon as the victim is removed from the carbon monoxide-containing environment and decreases more quickly if oxygen therapy is instituted, a normal COHb saturation in the emergency room does not exclude significant prior carbon monoxide exposure.

The half-life of COHb in a person breathing room air is approximately 250 minutes, but this declines to 40 minutes when 100 percent oxygen is breathed.[32] For this reason, fire victims should receive continuous supplemental high-flow oxygen therapy from the time they are removed from the fire until their COHb saturation is documented to be normal. Patients with severe carbon monoxide intoxication may require intubation and mechanical ventilation to allow delivery of 100 percent

TABLE 2.—*Increase in Blood Carboxyhemoglobin (COHb) Level During Heavy Exertion**†*

Carbon Monoxide Concentration		*Increase in COHb Saturation After Brief Carbon Monoxide Exposure (%)*			
(ppm)	*(%)*	*10 sec*	*30 sec*	*60 sec*	*120 sec*
1,000	(0.1)	0.2	0.6	1.3	2.5
10,000	(1.0)	2.5	7.5	15.0	30.0
20,000	(2.0)	5.0	15.0	30.0	60.0
50,000	(2.0)	12.5	38.0	75.0	..
100,000	(10.0)	25.0	75.0	..	..

*"Heavy exertion" = Labor demanding an alveolar ventilation rate of 30 liters/minute.
†Adapted and reproduced with permission from Stewart et al.[33]

TABLE 3.—*Signs and Symptoms at Various Concentrations of Carboxyhemoglobin (COHb)**

Concentration of COHb (%)	*Signs and Symptoms*
0-10	None in previously healthy patients
10-20	Tightness across the forehead, mild headache
20-30	Throbbing headache
30-40	Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting and collapse
40-50	Same as above, with greater possibility of collapse, syncope, and increased respiratory and pulse rates
50-60	Syncope, increased respiratory and pulse rates, coma, convulsions, and Cheyne-Stokes respirations
60-70	Coma, convulsions, cardiorespiratory compromise, death
>70	Death

*Modified from Schulte.[34]

oxygen and effective elimination of carbon monoxide. Hyperbaric oxygen and controlled hypothermia have also been advocated but are seldom available in time to be beneficial.[38]

Other Agents

Carbon monoxide intoxication may occur alone or be compounded by inhalation of other systemically active substances. Both carbon monoxide and cyanide were found in the blood of 39 of 53 persons who died in house fires.[39] In the remaining 14, only carbon monoxide was detected. In studies using rats, a reactive phosphate fire retardant has been shown to cause systemic toxicity that manifests as abnormal behavior, seizures and death, if the treated material continues to burn.[40]

Local Respiratory System Effects

Smoke inhalation victims are at risk of exposure to numerous other agents that cause not only systemic toxicity but also local damage to the respiratory system. Injury may occur at (1) the level of the upper airways, including the mouth, pharynx and larynx, (2) the lower airways, including the trachea, bronchi and bronchioles and (3) the pulmonary parenchyma. Local injury is a major cause of morbidity and mortality in burn patients. In one study 29 of 66 burn patients with local respiratory tract injury died as a result of respiratory complications.[21] In another study 94 of 197 patients died, and 69 of the deaths resulted from respiratory tract involvement.[22] The overall mortality of those burn patients with inhalational injury is two to four times that of patients without such injury.[20,41-44]

Diagnosis

There are no uniform criteria for making the diagnosis of local respiratory system damage or direct inhalational injury. Numerous clinical criteria have been described. The classic triad required for diagnosis of direct inhalational injury is the presence of thermal burns involving the face, particularly the mouth and nose, singed nasal vibrissae and a history of the burn injury being sustained in a closed space.[23] A combination of the above criteria, plus a history of inhalation of large amounts of smoke, the presence of carbonaceous sputum and clinical symptoms of wheezing, hoarseness or dyspnea have also been used.[21] Still other clinicians have required only that the patients have been exposed to the smoke of a fire and have sustained no more than minor cutaneous burns.[45] Patients with direct inhalation injury, as reported in the literature, vary considerably in the extent of the body surface area that has been burned, but most have sustained cutaneous burns.

The incidence of direct injury diagnosed by these various clinical criteria varies from 2.9 percent to 15.1 percent of all burn patients.[21,22,24,41] Clinical findings mirror the diagnostic criteria and include facial and oropharyngeal burns, cyanosis, dyspnea, tachypnea, cough, hoarseness, wheezes, rales, rhonchi and the presence of carbonaceous sputum.[21,23] In these clinically diagnosed patients, positive findings on auscultation of the chest at the time of admission[23] or development of cough, dyspnea, cyanosis, rales, rhonchi or wheezes within the first few hours after injury[24]—before significant fluid resuscitation occurred—were identified as poor prognostic indicators.

In the past decade much effort has been made to improve the accuracy of the early diagnosis of direct inhalation injury in burn patients. Radioactive xenon lung scanning and bronchoscopy have been studied the most. Inhalation injury is diagnosed by xenon lung scan if a prolonged length of time is taken to clear the xenon from the lungs or if segmental retention of the isotope occurs after intravenous administration.[43,44] Such abnormalities on scanning are associated with a history of injury in a closed space, findings of hoarseness and wheezing on physical examination, the presence of carbonaceous sputum, subsequent development of abnormalities found on roentgenograms of the chest and increased mortality. Poor correlation is seen with the presence of facial or intraoral burns. False-positive findings of xenon lung scans may occur in patients with preexisting bronchitis, bronchiectasis, asthma or pneumonia.[43] Inhalation injury is diagnosed bronchoscopically when laryngeal or tracheobronchial erythema or edema, carbonaceous material or bronchorrhea is seen.[42,46] With more extensive injury, blisters, ulceration, hemorrhage or necrosis may be present.

The incidence of direct inhalation injury in burn patients varies from 30 percent to 60 percent when these two specialized diagnostic techniques are used.[20,42-44] However, diagnosis of direct inhalation injury by either of these two methods does not specifically predict the subsequent occurrence of pulmonary complications. While respiratory problems developed in less than 10 percent of burn patients with negative findings on bronchoscopy or xenon scans, in 25 percent to

50 percent of patients with positive findings, clinically significant pulmonary complications did not develop.[20,42-44]

Roentgenograms of the chest, arterial blood gas measurements, cytologic evaluation of sputum and pulmonary function tests have also been examined as a means of making an early diagnosis of direct inhalation injury. Findings on chest x-ray films at admission are usually normal.* Determination of arterial blood Po_2, obtained while the patient is breathing room air at the time of admission, may also be within normal limits.[42] A progressive increase in the alveolar-arterial oxygen gradient was useful in predicting subsequent pulmonary dysfunction in a retrospective review of the clinical course of 41 burn patients considered at risk of inhalation injury.[49] The alveolar-arterial oxygen gradient measured while breathing 100 percent oxygen at 12 to 24 hours after injury was greater in patients whose condition subsequently deteriorated, and it increased even further 48 to 72 hours after the injury. Cytologic evaluation of expectorated ciliated epithelial cells has been suggested in one preliminary report to be a noninvasive way to diagnose damage to the airways.[50] Pulmonary function studies carried out in small numbers of patients with acute smoke inhalation have shown increased work of breathing, increased airways resistance, decreased flow rates and decreased lung compliance.[48,51-53]

At present the diagnosis of local respiratory system injury from smoke inhalation is a clinical one despite the variable clinical diagnostic criteria. When present, the diagnosis is supported by abnormal arterial blood gas measurements, findings of chest roentgenograms or pulmonary function tests. The routine use of bronchoscopy for direct visualization of the lower airways or of xenon lung scans is not recommended. The one critical exception to the predominantly clinical evaluation of patients with direct inhalation injury is that the patency of the upper airway must be evaluated by direct observation.

Upper Airway Obstruction

Obstruction of the upper airway from extreme laryngeal edema is a potentially lethal complication of smoke inhalation that requires prompt diagnosis and aggressive management. In one study, 8 of 33 patients with direct inhalation injury required intubation for laryngeal edema and obstruction of the airway,[20] and 5 of 15 required intubation for either obstruction of the airway or progressive respiratory insufficiency in another.[42] Edema and obstruction of the upper airway usually develop within 24 hours of injury.[51] Early clinical signs include tachypnea, hoarseness and stridor; however, these signs do not distinguish patients who progress, often precipitously, to complete airways obstruction.[20,54]

Direct visualization of the larynx can be used to identify patients at risk of developing progressive obstruction of the upper airway. Wanner and Cutchavaree[54] used fiberoptic transnasal laryngoscopy to examine 12 of 15 patients with a history of smoke inhalation and respiratory signs and symptoms of hoarseness, cough, dyspnea, wheezing and rhonchi. Of these, 3 had severe laryngeal edema; in 2 of these 3, the condition progressed to require intubation for maintenance of an adequate airway. The remaining nine patients displayed lesser degrees of edema and inflammation and showed no evidence of progressive airway obstruction.

Early direct visualization of the larynx should be done in any patient with a history of smoke inhalation and respiratory signs and symptoms including dyspnea, cough, hoarseness, stridor, tachypnea, wheezing or rhonchi. Patients with severe edema should undergo intubation.[54,55] If the initial examination of the larynx does not suggest the need for intubation but the patient's condition deteriorates, repeat visualization of the larynx is indicated. The edema usually subsides within three to five days of injury, and unless further complications are present, extubation is then possible.[20,55,56]

Long-term Complications

The long-term complications of local respiratory system injury are not well characterized. In the Cocoanut Grove disaster, 27 survivors who sustained mild-to-severe involvement of the respiratory tract were asymptomatic, with normal findings on physical examination and roentgenograms of the chest nine months later.[4] Tracheal stenosis, bronchial stenosis, bronchiolitis obliterans, endobronchial polyposis and bronchiectasis have been occasionally reported.[21,57-60]

Management

Initial Evaluation

An approach to the initial evaluation of a patient with smoke inhalation is summarized in Table 4. Historical details of the inhalation ex-

*20-22, 24, 25, 42, 47, 48

posure allow the types and degree of injury to be anticipated. Preexisting cardiopulmonary or central nervous system illness will affect the patient's ability to tolerate the additional insult.

During the physical examination positive findings on auscultation of the chest or findings suggestive of obstruction of the upper airway should be sought as should evidence for cardiac or neurological dysfunction. Quantification of the extent of the burn and the presence of other injuries such as lacerations or fractures should also be noted.

Determination of COHb saturation should be carried out in every patient. The need for additional studies will be suggested by the history and clinical findings. The clinical diagnosis of local respiratory system injury is supported by associated abnormal arterial blood gas measurements and abnormal findings of chest roentgenograms and bedside pulmonary function tests. At the initial evaluation, the question of the patency of the patient's upper airway must be raised and the need for laryngoscopy considered.

TABLE 4.—*Initial Evaluation of a Smoke Inhalation Victim*

History: Ascertain
What were the circumstances of injury—closed area?
What material was burned?
Was there steam exposure?
What prior cardiopulmonary or CNS diseases are present?
What respiratory, cardiac, CNS symptoms are present?
Physicial examination: Observe for
Tachypnea
Facial, oropharyngeal, nasal hair burns
Hoarseness, stridor, wheezing, rales, rhonchi
Abnormal cardiovascular or neurological findings
Burn extent and other injuries
Laboratory: Obtain
Sputum specimen for carbonaceous material
Arterial blood gas measurements: Po_2, Pco_2, pH, COHb
Roentgenogram of chest
Electrocardiogram
Bedside pulmonary function tests: FEV_1/FVC
Laryngoscopy

CNS = central nervous system; COHb = carboxyhemoglobin saturation; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; Pco_2 = partial pressure of carbon dioxide; Po_2 = partial pressure of oxygen.

TABLE 5.—*Treatment of Smoke Inhalation*

At the scene of the fire
Maintenance of an adequate airway
Delivery of supplemental oxygen therapy
Provision of basic resuscitative measures
In the hospital
Assessment of airway patency
Laryngoscopy
Intubation
Maintenance of adequate pulmonary toilet
Delivery of humidified supplemental oxygen therapy
Use of bronchodilator therapy
Preparation of sputum Gram stain
Replacement of fluid losses

Treatment

The treatment of patients with inhalation injury is, for the most part, supportive and is outlined in Table 5. Specific variations reflect the toxicology of any inhaled substances that are identified. At the scene of the fire, maintenance of an adequate airway and administration of oxygen to treat presumed carbon monoxide intoxication are priorities along with other basic resuscitative measures.

Once at the hospital, assessment and treatment of the patient's injuries from smoke inhalation, thermal burns and other trauma continue. Severe laryngeal edema observed on direct visualization of the larynx is an indication for intubation. Attention should be focused on maintaining adequate pulmonary toilet and on delivery of humidified supplemental oxygen. Bronchodilator therapy is useful in treating the bronchospastic component of the airway obstruction. Because of the increased risk of pneumonia, daily sputum Gram stains should be done and findings correlated with the patient's clinical course. Prophylactic antibiotic therapy has no demonstrated role in patients with direct inhalation injury.[11,22,24]

The use of corticosteroids in the treatment of direct inhalation injury has been recommended by some, but the results of most studies do not support their use. In one controlled study of rats exposed to white pine smoke, methylprednisolone or dexamethasone improved survival.[3] However, dexamethasone had no effect on mortality or morbidity in a prospective randomized study of 30 humans.[61] Furthermore, a partially randomized study of 33 patients treated with methylprednisolone showed a threefold increase in the occurrence of pneumonia and bacteremia and a fourfold increase in mortality.[20] At a consensus development conference on supportive therapy in burn care in 1978, the recommendation was made that corticosteroid therapy should not be used in the treatment of direct inhalation injury.[62]

Judicious administration of fluids should be monitored by following blood pressure and urine output. Measurement of pulmonary capillary wedge pressure and cardiac output should be used

to guide fluid therapy in patients with hemodynamic compromise.[63,64]

After discharge, subsequent evaluation with measurement of arterial blood gases, pulmonary function studies and bronchoscopy should be guided by the patient's clinical status; these are not routinely recommended.

Admission Guidelines

Widely accepted guidelines are not available to aid clinicians in deciding which patient with smoke inhalation injury should be admitted to hospital. In Table 6, several findings are listed that, if present, should suggest the need for further observation in an inpatient setting. Patients whose initial assessment does not indicate the need for admission should be reassessed over the next 6 to 12 hours. Deterioration of clinical status or laboratory values is an indication for hospital admission.

TABLE 6.—*Guidelines for Admission of Patients With Smoke Inhalation Injury*

Presence of significant thermal or other injuries
Symptoms, signs or laboratory evidence of cardiac or CNS dysfunction
Physical examination findings of: tachypnea, hoarseness, stridor, wheezing, rales, rhonchi
COHb saturation >15% to 20% on arrival
Abnormal results of chest roentgenogram, arterial blood gas measurement, pulmonary function tests
Deterioration in clinical status, arterial blood gases, pulmonary function tests

CNS = central nervous system; COHb = carboxyhemoglobin

TABLE 7.—*Guidelines for Fire Safety in High-Rise Buildings**

Know the location of two exit stairs near your room.
Count the number of doors between you and the exit stair so that the exit can be found by feel in a smoky and dark hallway.
Never use the elevator in the event of a fire.
Know where the room key is and always take it with you when you leave the room so that you will not be locked out.
Report any fire you discover to the building personnel and to the fire department.
Avoid breathing the smoke.
Seal cracks around the door of your room and try to close any air ventilators in the room.
Feel the door before opening it. If it is hot, keep it closed.
Do not walk into smoke. Stoop down or crawl.

*Adapted from the pamphlet "If You Work . . . If You're Staying or Live in a High-Rise You Can Survive! But It's Up to You," available from the Community Relations Section, Fire Prevention Bureau, Los Angeles County Fire Department.

Prevention

It has been stated that an ounce of prevention is worth a pound of cure. The use of smoke detectors provides an early warning system that is lifesaving. Knowing in advance what to do if trapped in a burning building or smoked-filled area may mean the difference between survival and death. Table 7 summarizes warnings and guidelines issued by the Los Angeles County Fire Department in an effort to improve survival in fires in high-rise buildings.

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